Hypoxemia

Causes of Hypoxemia

<table>
<thead>
<tr>
<th>Causes of Hypoxemia</th>
<th>P_aCO2</th>
<th>A-a Gradient</th>
<th>DLCO</th>
<th>Corrects w/ 100% F_iO2?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low F_iO2</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Yes</td>
</tr>
<tr>
<td>Hypoventilation</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>Yes</td>
</tr>
<tr>
<td>Diffusion Impairment</td>
<td>Normal</td>
<td>↑</td>
<td>↓</td>
<td>Yes</td>
</tr>
<tr>
<td>Shunt</td>
<td>Normal</td>
<td>↑</td>
<td>Normal</td>
<td>No</td>
</tr>
<tr>
<td>V/Q Mismatch</td>
<td>Normal / ↑</td>
<td>↑</td>
<td>Normal</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Shunt: perfusion without ventilation (V/Q=0); see ↓pO2
Dead Space: ventilation without perfusion (V/Q=∞); see ↑pCO2

Equations

**Alveolar-arterial (A-a) Gradient**

\[ P_{(A-a)O_2} = P_{aO_2} - P_{aO_2} \]

**Alveolar Gas Equation**

\[ P_{aO_2} = F_iO_2 \times (P_{atm} - P_{H2O}) - (P_{aCO_2} / 0.8) \]

\[ = 0.21 \times (760 - 47) - (40 / 0.8) \]

\[ = 100 \text{ mm Hg} \]

Normal A-a Gradient:

- < 10 mm Hg (F_iO_2 = 0.21)
- < 60 mm Hg (F_iO_2 = 1.00)
- < (age / 4) + 4
- a/A ratio > 0.75

Normal P_aO_2:

- 103 - age / 3

1. Low F_iO_2
   - Altitude
   - Hypoxic F_iO_2 gas mixture

2. Hypoventilation
   - Drugs (opioids, BZDs, barbiturates)
   - Chest wall damage
   - Neuromuscular diseases
   - Obstruction (e.g. OSA, upper airway compression)

3. Diffusion Impairment
   - Increased diffusion pathway (e.g. pulmonary edema, fibrosis)
   - Decreased surface area (e.g. emphysema, pneumonectomy)
   - Decreased rate of O_2-Hb association (e.g. high CO, anemia, PE)
Causes of Hypoxemia

4. **Shunt** (i.e. perfusion w/o ventilation; V/Q = 0)
   - Congenital (e.g. ASD, VSD, PDA), or AVM
   - ARDS, pneumonia, atelectasis

5. **V/Q Mismatch**
   - Often multifactorial
   - COPD, ILD, PE
   - Decreased CO (e.g. MI, CHF)

6. **Mixed Process**
   - Hypoxemia is often due to multiple causes.
   - Example: A tourist with COPD is visiting Denver, overdoses on heroin, now s/p MVA with chest wall trauma, pulmonary hemorrhage, Hct = 15%, and LV contusion. What is the cause of hypoxemia?

Hypoxemia in the OR

**Take a systematic approach to the diagnosis and treatment of hypoxemia in the OR!**

**Suggestion:** Alveoli → Machine

1. **Listen to the lungs**
   - Atelectasis
   - Pulmonary edema
   - Bronchoconstriction
   - Mucus plug or secretions
   - Right mainstem ETT
   - Pneumothorax
   - Esophageal intubation

2. **Check ETT**
   - Cuff deflation
   - Kinked/bitten ETT
   - Extubation

3. **Check circuit**
   - ETT disconnect
   - Circuit disconnect

4. **Check machine**
   - Inspiratory & expiratory valves
   - Bellows
   - Minute ventilation
   - F\(_{2}\)O\(_{2}\)
   - Pipeline & cylinder pressures

5. **Check monitors to confirm (you will probably do this 1st!)**
   - Pulse oximeter waveform
   - Gas analyzer

Management of Hypoxemia

Assuming proper oximeter function, placement, and waveform:

- Place patient on 100% O\(_{2}\).
- Perform recruitment maneuver and/or add PEEP.
- Confirm ETT placement by auscultation, bilateral chest rise, and FOB if necessary.
- Suction airway.
- Consider cardiovascular causes and restore volume, RBCs and/or cardiac output.
O₂-Hb Dissociation Curve

Useful "anchor" points:

<table>
<thead>
<tr>
<th>SO₂</th>
<th>PO₂ (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>50%</td>
<td>27</td>
</tr>
<tr>
<td>75%</td>
<td>40</td>
</tr>
<tr>
<td>97%</td>
<td>100</td>
</tr>
</tbody>
</table>

Note: P₅₀ ≈ 27 mm Hg

O₂-Hb Curve Shifts

Right Shift (lower affinity for O₂ = increased unloading at tissues)
- Acidosis
- Hyperthermia
- Hypercarbia
- Increased 2,3-DPG
- Sickle Cell Hb
- Pregnancy
- Volatile anesthetics
- Chronic anemia

Left Shift (higher affinity for O₂ = decreased unloading at tissues)
- Alkalosis
- Hypothermia
- Hypocarbia
- Decreased 2,3-DPG
- CO-Hb
- Met-Hb
- Sulf-Hb
- Fetal Hb
- Myoglobin

Factors Affecting Tissue Oxygenation

- Hb concentration
- O₂ Saturation
- Cardiac Output
- O₂ Consumption
- O₂-Hb Affinity (P₅₀)
- Dissolved O₂ in plasma (little effect)

See “Equations” for a mathematical explanation of these factors.

Arterial O₂ Content
\[ C_aO_2 = O_2-Hb + \text{Dissolved O}_2 \]
\[ = (Hb \times 1.36 \times S_oO_2/100) + (P_oO_2 \times 0.003) \]
\[ = (15 \times 1.36 \times 100) + (100 \times 0.003) \]
\[ ≈ 20 \text{ cc } O_2/dl \]

Mixed Venous O₂ Content
\[ C_vO_2 = O_2-Hb + \text{Dissolved O}_2 \]
\[ = (Hb \times 1.36 \times S_vO_2/100) + (P_vO_2 \times 0.003) \]
\[ = (15 \times 1.36 \times 75) + (40 \times 0.003) \]
\[ ≈ 15 \text{ cc } O_2/dl \]
Equations

**O₂ Delivery**
\[ \text{DO}_2 = \text{CO} \times \text{C}_a\text{O}_2 \]
- \[= 5 \text{L/min} \times 20 \text{cc O}_2/\text{dl} \]
- \[= 1 \text{L O}_2/\text{min} \]

**O₂ Consumption (Fick Equation)**
\[ \text{VO}_2 = \text{CO} \times (\text{C}_a\text{O}_2 - \text{C}_v\text{O}_2) \]
- \[= 5 \text{L/min} \times 5 \text{cc O}_2/\text{dl} \]
- \[= 250 \text{cc O}_2/\text{min} \]

**O₂ Extraction Ratio**
\[ \text{ER}_{O2} = (\text{VO}_2 / \text{DO}_2) \times 100 \]
- \[= 250 / 1000 \]
- \[= 25\% \text{ (normal 22-30\%)} \]

Other Concepts

**Diffusion Hypoxia** = low PₐO₂ as a result of breathing air, in combination with the washout of N₂O into the alveoli, upon termination of an anesthetic.

**Absorption Atelectasis** = the tendency for airways to collapse if proximally obstructed; poorly soluble N₂ normally stents alveoli open, but patients on 100% O₂ have greater tendency toward atelectasis.

**Bohr Effect** = a property of Hb in which increasing CO₂, temperature, and acidosis promote decreased O₂-Hb affinity (i.e. right-shift of O₂-Hb curve).

**Haldane Effect** = a property of Hb in which O₂ promotes dissociation of CO₂ from Hb to the plasma (e.g. as when venous blood enters the lungs).

References

